REMARKS

By way of the above amendment, Applicants hereby cancel claims 1-7 and 9-11. Claim 8 has been amended solely to incorporate subject matter from canceled claim 5 from which it depended. New claims 12-16 are added. Support for the amendment can be found throughout the specification as filed, for example, at page 78, line 24 - page 88, line 16. No new matter has been added. The above amendment is not to be construed as acquiescence to the stated grounds for objection/rejection and is made without prejudice to prosecution of any subject matter modified and/or removed by this amendment in a related divisional, continuation and/or continuation-in-part application. Reconsideration of the claims is respectfully requested in view of the above amendment and the following remarks.

***Priority***

The Action alleges that Applicants' claim to an earlier effective filing date only goes through US non-provisional application 09/285,479 ('479) filed 4/2/1999. The Action goes on to allege that the '479 application does not provide adequate written description of administering to a patient of composition comprising an antibody for SEQ ID NO:161. Accordingly, the Action accords to claim 8 the priority date of 12/17/1999.

As an initial matter, Applicants respectfully note that Applicants' claim to an effective earlier filing date as outlined on the ADS as filed at page 5 also includes claims to US non-provisional applications 09/221,107 filed 12/22/1998, 09/123,912 filed 07/27/1998 and 09/040,802 filed 3/18/1998. Additionally, Applicants respectfully traverse the determination of priority and submit that support for claim 8 and the newly added claims can be found as far back as US non-provisional application 09/221,107 ('107) filed 12/22/1998. In particular, Applicants direct the Office to Example 3 at pages 37-39 of the '107 application where contigs 17, 19, and 24 are described as being overexpressed in head and neck and lung squamous tumors with no or very low expression in normal lung tissue and some low or moderate expression in a variety of other normal tissues. At the second to last paragraph on page 39 of the '107 application, it is clearly stated "Also, the full-length cDNA sequence incorporating Contigs 17, 19, and 24, referred to as L762P, is provided in SEQ ID NO: 160, with the corresponding predicted amino

acid sequence being provided in SEQ ID NO: 161." Moreover, for example, at page 25, the '107 application clearly describes the use of antibodies as therapeutic agents. Accordingly, the skilled artisan would readily appreciate upon reading the '107 disclosure that the sequence of SEQ ID NO:161 is shown to be overexpressed in lung tumor tissue as compared to expression in normal lung and other normal tissues and that antibodies that specifically bind to this polypeptide can be used as therapeutic agents in the context of lung cancer. Accordingly, Applicants respectfully submit that claim 8 and the newly added claims 12-16 are entitled to the earlier effective filing date of 12/22/1998, the filing date of the '107 application. A supplemental ADS is included herewith reflecting this priority claim. Reconsideration is respectfully requested.

***Claim rejections under 35 U.S.C. § 102(e)***

Claim 8 stands rejected under 35 U.S.C. § 102(e) as allegedly anticipated by Holroyd *et al.* (US Patent 6,576,434) or Pauli *et al.* (US Patent 6,309,857) as evidenced by Abbas *et al.* (page 393, column 2, section antibodies, Cellular and Molecular Immunology, 4th edition, published by W.B. Sauders Co., 2000). In particular, the Action contends that Holroyd *et al.* discloses pharmaceutical compositions comprising an antibody to a protein that is 99.6% identical to the recited protein set forth in SEQ ID NO:161 and methods of using same for treatment. Further, the Action contends that Pauli *et al.* discloses a pharmaceutical composition comprising an antibody to a protein specifically expressed in lung, that is 99.7% identical to the protein set forth in SEQ ID NO:161. The Action additionally asserts that Pauli discloses a method for preventing lung-metastatic tumor spreading by administering such an antibody. In view of the teachings of Abbas *et al.*, the Action concludes that the present invention is anticipated.

Applicants respectfully traverse the rejection on the following grounds. As an initial matter, Applicants note that the Holroyd *et al.* patent cannot be used as prior art against the present invention under 35 U.S.C. § 102(e). In particular, Holroyd *et al.* is a US Patent of an International Application that was filed before November 29, 2000. As such, the 102(e) date is the § 371(c)(1), (2), (4) date, which is February 13, 2001 and not the US provisional filing date

of March 3, 1998 referred to on the face of the patent. Accordingly, this reference is not prior art to the present invention. Withdrawal of the rejection is respectfully requested.

Concerning Pauli *et al.*, Applicants submit that the teachings set forth in this reference do not in any way anticipate the presently claimed invention. In particular, Pauli *et al.* teaches the identification of nucleotide sequences encoding mammalian calcium activated chloride channel-adhesion molecules. One of these molecules (*e.g.*, SEQ ID NO:32) has 99.7% identity to the recited protein of SEQ ID NO:161. The Action alleges that Pauli *et al.* teaches that this similar sequence is specifically expressed in lung. In fact, Pauli *et al.* teaches at Column 14, lines 27-32 that the hCLCA2 mRNA was not detected in lung by Northern blot hybridization. This sequence was detected in lung, trachea and mammary gland using RT-PCR, a more sensitive technique. Thus, the authors conclude that this "[suggests] a significantly lower expression level in the lung."

The Action also contends that Pauli *et al.* discloses using an antibody to the hCLCA2 protein to prevent lung-metastatic tumor spreading. Applicants respectfully disagree with the Action's interpretation of the teachings of Pauli *et al.* The cited reference describes using bovine recombinant and wild-type Lu-ECAM-1 in a cell adhesion assay to test adhesion of the bovine molecules to the murine B16-F10 metastatic melanoma cell line (Column 18, lines 31-41). In this experiment, recombinant bovine Lu-ECAM-1 was better able to adhere to the murine metastatic melanoma cell line than the wild-type bovine Lu-ECAM-1. The interaction of wt-Lu-ECAM-1 was almost completely blocked by an antibody specific for the bovine Lu-ECAM-1. This antibody only partially blocked the recombinant bovine Lu-ECAM-1 interaction. From this experiment, the authors speculate by way of a prophetic example (Example 9, cited by the Action) that administering an antibody raised against bovine Lu-ECAM-1 can be used to block "lung-metastatic" tumor cells. SEQ ID NO:32, the sequence disclosed by Pauli *et al.* that has the highest identity to the presently recited SEQ ID NO:161, has only 63.7% identity to the bovine Lu-ECAM-1 protein (see Table 2, first row, last column). Further, there is simply no teaching by Pauli *et al.* that the monoclonal antibody specific for the bovine Lu-ECAM-1 protein used in the adhesion experiments binds to any of the human sequences, let alone one that has only 63.7% identity to the bovine protein and no indication that

this antibody can block adhesion of cells expressing the human protein of SEQ ID NO:32 (or the presently recited protein of SEQ ID NO:161) to metastatic melanoma cells. Thus, Pauli *et al.*, as outlined above, teaches nothing regarding the expression of the protein set forth in SEQ ID NO:161, or even similar proteins, in lung cancer as compared to normal lung tissue nor does this reference teach the use of antibodies specific for the protein of SEQ ID NO:161 for increasing an immune response. The general antibody teachings of Abbas *et al.* do not remedy the shortcomings of Pauli *et al.* Accordingly, Applicants submit that the cited references do not anticipate the presently claimed invention. Reconsideration and withdrawal of the rejection is respectfully requested.

In view of the above amendments and remarks, the claims are now believed to be in condition for allowance. A good faith effort has been made to place the application in condition for allowance. However, should any further issue require attention prior to allowance, the Examiner is requested to contact the undersigned at 206-622-4900 to resolve same.

The Director is authorized to charge any additional fees due by way of this Amendment, or credit any overpayment, to our Deposit Account No. 19-1090.

Respectfully submitted,

SEED Intellectual Property Law Group PLLC

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